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ACUTE ANTERIOR POLIOMYELITIS

A Brief Review

K. K. GREGORY, M.D.

The mortality rate in acute anterior poliomyelitis in an epidemic is between seven and ten per cent. There are many diseases in which the mortality rate is above this and yet poliomyelitis is still one of the most dreaded of diseases. The loss of a loved one in death is cause for great sorrow, but time soon comforts and heals the aching heart. In poliomyelitis, the deformed, maimed bodies and the life-long helplessness of the victims are a constant source of heart-ache and pity, to say nothing of the economic burden both on the family and the community.

It is little wonder, then, that fear grips the hearts of parents when the disease appears in their midst. In an epidemic, this often develops into hysteria. Consequently, this dreaded and so-called mysterious disease has aroused a tremendous amount of interest among scientists as well as among laymen. Extensive studies and research have been undertaken to learn more about the disease and a large amount of money has been expended for the rehabilitation of its victims. Great progress has been made by these efforts, yet much remains to be accomplished. There have been many false leads and false hopes. Nevertheless, notable contributions have been made and among these, the latest, and to my mind the most important, at least from the patient's standpoint, is Miss Kenny's treatment of the symptoms of acute poliomyelitis. Miss Kenny is of the opinion that poliomyelitis will lose much of its damaging effects when her treatment is universally practised. The disease will then be relegated into a minor position in much the same way as typhoid fever has become less important.

History

Poliomyelitis is not a new disease. Records of human bones and ancient engravings indicate unmistakably that the disease existed long before recorded history. The first epidemic of which we have record occurred in England in 1835, as reported by Badlam. In 1840, Jacob Heine, an orthopedic surgeon in Germany, gave us the first clear description of the disease in the acute stage. Fifty years later (1890), Medin of Sweden distinguished the bulbar, ataxic, encephalitic, polyneuritic, and spinal types. He also recognized the disease as occurring both in endemic and epidemic form. Calverly, in 1894, and Wichman, between 1905 and 1911, recognized the non-paralytic or abortive cases, and the latter showed that the disease was infectious and that healthy carriers played an important part in the spread of the disease.

Poliomyelitis is world-wide in distribution and while no region is excepted, it is more common in Northern Europe, Northern United States, Canada, New Zealand, and Australia. Unlike most infectious diseases which are prevalent in cold weather, poliomyelitis reaches high incidence in the late summer and early fall months. However, severe epidemics of the disease have occurred in Norway and Sweden during the winter months. In the United States, the epidemic in 1916 was the greatest, with a total reported cases of 27,363. Epidemics of lesser extent have occurred every two to six years since 1921. The total number of reported cases up through 1937 was 179,369. While this number is not large when compared with other infectious diseases, it is important for it accounts for thirty to sixty per cent of the cripples in the country.

Etiology

The etiology of poliomyelitis is now generally accepted to be a filterable virus. Landsteiner of Vienna is credited with performing the first successful transfer of the disease to an experimental

animal, the rhesus monkey, by the intraperitoneal injection of a cord suspension from a fatal human case in 1908. This work was subsequently confirmed and the etiologic agent found to be filterable through the finest porcelain. Other causal agents were suggested but only the alpha-hemolytic streptococci advocated by Rosenaw, and the "globoid bodies" of Flexner and Noguchi were seriously considered but they have now been satisfactorily excluded by competent investigators in the field. It has been found that there are several different strains of the poliomyelitis virus, probably as many as there are of pneumococci, each with differing degrees of virulence, adaptability, and immunilogic properties.

While much has been learned about the poliomyelitis virus, progress has been necessarily slow. This is due primarily to the inability of the virus to grow either on artificial media or in the usual laboratory experimental animals. Up to three years ago, monkeys, chimpanzees, and living human nervous tissues were the only means for a successful cultivation of the virus. This was a distinct handicap. However, Armstrong of the United States Public Health Service, reported in 1939 that he was able to transfer the Lansing strain of virus to the cotton rat, and subsequently to white mice and then back to monkeys. This discovery was received with enthusiasm by his fellow-workers. It is now possible for the research worker not only to use different strains of the poliomyelitis virus but also to use cotton rats or white mice for research work instead of the hard-to-get and expensive monkeys or chimpanzees.

Neutralization Test

It was found, early in experimental poliomyelitis, that the blood of monkeys which had recovered from the disease possessed specific antibodies which not only neutralized the virus in vitro but also protected well monkeys against intracerebral injection of the virus. Similar immune substances were also found in recovered human patients. This lead to the hope that convalescent human serum might be effective for prophylaxis and treatment. The convalescent serum was given a good trial throughout the country but the initial high hopes fell when statistics showed that there was no difference between the treated and the control cases. Consequently, the use of convalescent human serum has been abandoned by most clinicians. Aycock, who was the first to advocate its use, was the first to abandon it.

Neutralization test studies have been extended in recent years and these have shown that a high percentage of normal human adults, even in tropical and subtropical regions where clinical poliomyelitis cases are rare, carry specific antibodies in their blood. It has also been found that a higher percentage of adults in cities possess specific antibodies than those in rural districts, and that young children have little or none at all. These findings have been interpreted as the result of a subclinical attack of the disease, perhaps in much the same way as subclinical attacks of scarlet fever or diphtheria develop active immunity.

This explanation has not been accepted by all investigators, however, for there are several discrepancies in it. It is known that patients who have recovered from clinical poliomyelitis have, in many instances, a lower titer in the neutralization test than others who have not had the disease. Furthermore, it has been hard for some to accept the subclinical attack of poliomyelitis as the explanation for the high percentage of positive neutralization tests among people in the tropical and subtropical countries where clinical cases of the diseases are rare. This group of authorities has advanced the theory that the presence of immune substances in the blood of adults is the result of maturation and experiments have been undertaken to support this contention. However, the question is not settled. If this latter theory proves eventually to be correct, it may change our entire conception of immunity.

Active Immunity

Active immunity has been obtained in the experimental monkey by the inoculation of either the attenuated virus or sublethal doses of virulent poliomyelitis virus. Brodie and Park, using formalized virus, which had been found non-infectious for monkeys, inoculated 2,300 children. Kolmer, employing a mixture of virus in sodium ricinoleate, which was admittedly infectious for monkeys, inoculated 10,725 children. Three of the children immunized by Brodie and Park and nine of those immunized by Kolmer developed poliomyelitis. This was unfortunate, for while there was no definite evidence that the vaccines had caused the disease in these twelve cases, the circumstances under which they occurred, and the

fact that they developed in localities where poliomyelitis was not present at the time, pointed to the vaccine as the probable source of infection. This has ended, for the present at least, efforts to develop active immunity in the human by these methods.

Pathology

In interpreting the early symptoms of poliomyelitis on the basis of pathology, before the onset of paralysis, two questions are raised. First, is poliomyelitis a generalized infection involving primarily the lymphoid tissues and attacking the central nervous system only secondarily and rarely? Or, second, is the disease strictly an infection of the nervous system with the clinical symptoms the result of the damage done to it?

The first interpretation is the earlier and the one more commonly recognized. It has the support of many able investigators. It is believed that the generalized infection only occasionally involves the meninges and that subsequently the degeneration of the nerve cells in the anterior horn of the cord is secondary to the perivascular round cell infiltration and edema. Damage to the nerve cells is not complete, in many instances, and therefore many of them are restored to full function as soon as the edema subsides. This is the explanation for the prompt and spontaneous recovery of function in a supposedly paralyzed muscle or group of muscles often seen in the acute stage of the disease. In recent years, however, this interpretation of the pathology of poliomyelitis has lost many adherents because detailed experimental studies have shown that the virus attacks the nerve cells directly and early, before extensive perivascular cellular infiltration has taken place. It has also been found that damage to nerve cells is present even in the pre-paralytic stage of the disease. Therefore, the early symptoms of the disease, according to this idea, are due not to generalized infection but rather to infection of the central nervous system, especially the spinal cord.

The pathology in poliomyelitis is confined primarily to the anterior horn cells of the cord and motor nuclei of the medulla. It is believed that the virus has great affinity for the large nerve cells which it attacks and eventually destroys. Degeneration of these cells is probably not due to any toxic products created by the virus but probably is due to interference of the normal cell metabolism by the virus. Specifically just how this process of interference takes place is at present a subject for speculation.

Sources and Modes of Infection

Up to a few years ago, the nasopharynx was accepted, almost universally in this country, as the portal of entry as well as the portal of exit of the poliomyelitis virus. The virus has been recovered from the nasopharynx of acute, convalescent, and abortive cases, and also of contacts. Similar results have also been obtained in monkeys in all stages of the disease, following all routes of injection. It was also found that the rhesus monkey could be infected by the simple instillation of the virus intranasally and only with great difficulty by any other route. Further investigation revealed that the virus was present in the olfactory bulbs of monkeys infected by the nasal route, before it was present in other parts of the central nervous system.

To prove that this was the pathway of the infection, the olfactory bulbs were sectioned and it was found that the monkeys were no longer susceptible to infection by intranasal instillation of the virus. A further proof of this idea was the experiment by which the nasal mucosa was blocked by the application of zinc sulphate and it was successful in preventing the virus from entering the central nervous system after nasal inoculation. These experimental findings brought the conviction, and thereby the conclusion, that the nasopharynx was the portal of entrance as well as the chief channel for the exit of the virus. It was then only a matter of logic to assume that the disease in man was spread by the secretion from the nasopharynx of those harboring the virus and that the susceptibles became infected via the nasal mucosa. This theory of the source and mode of infection seemed foolproof and enjoyed universal acceptance until three or four years ago when it was seriously challenged by the gastro-intestinal portal idea.

The idea that the gastrointestinal tract acts as a reservoir for the virus is not new. Back in 1912, Kling and his co-workers reported the production of poliomyelitis in monkeys by the intraperitoneal injection of filtered washings from the intestines of acute, convalescent, and post-mortem human cases. But these workers failed to follow through and consequently their work was not accepted. The idea was revived in 1937 by the work of Harmon. He was able to isolate the virus from the stool of

human patients with fair success. It should be pointed out that Toomey of Cleveland had been for years one of the chief advocators of this theory. He was not only able to recover the virus from the stools and intestinal washings of patients, but he was also able to infect monkeys by introducing these materials into the intestinal tract by means of balloons which avoided contamination of the nasopharynx.

During the past few years, evidence has been accumulating to show that it is not only twice as easy to isolate the virus from the stools than it is from the nasopharynx of patients and carriers, but also the amount of the virus in the stool is great. It has been estimated that in a 50 gram stool, there are anywhere between 1,000 and 10,000 infective doses for the monkey. This great quantity of virus must enter sewage during an epidemic of the disease. Investigations have shown this to be true in six localities where the disease was prevalent at the time. That poliomyelitis is not a water-borne disease, in the true sense of the word, has been shown by twenty-five post-epidemic tests on sewage in four large cities. All of them were negative.

The gastrointestinal tract as the chief channel for the exit of the virus brings to the fore the suspicion long held by epidemiologists that beaches, rivers, and ponds are in some way connected with the disease because many epidemics have been traced along waterways. These could be polluted by the sewage or, in the case of ponds, by surface drainage. It has been found that patients or carriers may harbor the virus in the intestinal tract for months. This idea of the intestinal excreta being the chief means by which the disease is disseminated fits perfectly with the seasonal prevalence of the disease.

Now, with all these findings indicating the gastrointestinal tract as the chief source of the virus, the question is how the virus gets there. Is the virus, which is multiplying in the nasopharynx, swallowed and then excreted in the stool? Or, is the virus secreted in the gastrointestinal tract, having come from the central nervous system through the lymphatic system, and in like manner to the nasopharynx? Or, is the gastrointestinal tract both the portal of entry and exit, with the virus being brought there in the first place by the ingestion of infected milk, food, or water? If so, how does the virus gain entrance into the central nervous system? It is certainly not through the blood stream because the virus has never been demonstrated in it. Is it via the lymphatic system, as some investigators believe? There are those who contend that the virus is neurotropic and that it first gains entrance into the parasympathetic system, then through the axons of the vegetative nervous system into the spinal cord and brain, where it finally attacks the motor cells of the anterior horn and cortex of the brain. All of these are, however, only speculations at the present time.

Frequency in an Epidemic

Thus a large amount of information has been obtained from experimental work and while it has not solved the entire problem of poliomyelitis, it has helped explain some of the clinical features of the disease. The statement found in some of the old textbooks to the effect that it is seldom that more than one member in a family develops infantile paralysis is erroneous in view of our present knowledge of the disease. What the authors actually meant was that it is seldom that the disease paralyzes more than one member of a family. Poliomyelitis is a common disease during an epidemic. It is more widespread than we have been lead to believe by our former teachings. Clinically, the disease has been classified as abortive, preparalytic or non-paralytic, and paralytic, according to whether the symptoms are merely systemic or are either accompanied by or followed by paralysis.

Diagnosis

The diagnosis of poliomyelitis may be simple or difficult, depending on whether the patient shows evidence of paresis or muscle spasms. The non-paralytic case offers a problem in diagnosis which is very difficult to solve in the absence of an epidemic. However, symptoms of infection of the upper respiratory tract, together with headache, nausea and vomiting, diarrhea or constipation, and moderate fever, occurring in a patient in late summer or early fall are suggestive of poliomyelitis. Spasms of the muscles of the neck and back and an increase in the lymphocyte count of the spinal fluid are presumptuous evidence of poliomyelitis. If an epidemic is present, the diagnosis is more likely to be correct. The diagnosis of the very mild or abortive cases is difficult and often impossible even during an epidemic of the disease. However, under certain circumstances, such as a family epidemic

where a sibling has the disease with paralysis, a diagnosis of abortive poliomyelitis may be made of those in the family who complain of slight sore throat, headache, and slight malaise, with or without gastrointestinal disturbances. Such a diagnosis has been justified by numerous studies showing that in almost all of these cases, the virus has been recovered from the nasopharynx or stool or both, and that the neutralization tests have revealed abundant specific antibodies in their blood.

I shall not discuss the various paralytic types of the disease nor shall I discuss in detail the differential diagnosis except in two diseases, both of which are of virus origin, namely, lymphocytic choriomeningitis and Guillain-Barré Syndrome (polyradiculoneuritis with albumino-cytologic dissociation).

Lymphocytic choriomeningitis is characterized by symptoms and physical signs of meningeal irritation which are indistinguishable from those of purulent meningitis. The spinal fluid cell count is usually higher than that in poliomyelitis and the cells are all lymphocytes. If such a case occurred in the midst of an epidemic of poliomyelitis, it would be almost impossible to diagnose it as such.

Guillain-Barré Syndrome is a disease so much like poliomyelitis clinically that it is difficult to differentiate the two. There is pain, weakness, and incoordination of various muscles of the body. The most important factor in the diagnosis is the spinal fluid. In Guillain-Barré Syndrome, the cell count and sugar are normal, but the protein is high, as much as ten times the normal level. The sugar and protein in poliomyelitis are within normal limits.

Treatment

The treatment of poliomyelitis in the acute stage, as I have already alluded to in this paper, is symptomatic. Passive immunization with the convalescent human or horse serum has been given up by most clinicians. Active immunization has not been found feasible with present methods. Drugs, even the sulfonamides, have been tried without benefit.

Now I come to the treatment which is at present of much interest and of vital importance to all of us,—in fact, to the entire world—namely, Miss Kenny's treatment of acute poliomyelitis. Indeed, this is the main subject of our discussion tonight, and therefore I shall cease now and pass your attention to the next speaker who will tell you of the practical and more interesting part of this subject.

Charles V. Chapin Hospital

KENNY TECHNIQUE FOR INFANTILE PARALYSIS

Excerpts from the
Continuation Course (for Physicians)
at the University of Minnesota

William A. Horan, M.D.

My purpose here this evening is to present to you the Kenny Method of treatment for Infantile Paralysis as presented to us at the University of Minnesota, Center for Continuation Study. The University is in the City of Minneapolis and is the second largest University in the Country. The Center for Continuation Study, at the University of Minnesota, was opened in 1935 and since that time has harbored some 3500 physicians who have taken special courses of one kind or another. When we registered on September 20th, for a Course which was to extend from the 21st to the 26th, we were assigned to quarters that were quite homey, hotel-like, and designed to do away with the College idea of Dormitory Rooms. At the Center we were not restricted in any way, being permitted to come and go as we chose, and we were offered the use of the Dining Room. In fact, it was suggested that we use it, in order that we might discuss amongst ourselves the subject matter of the course. On the first floor of the building was a huge Lounge where we were to meet every afternoon for Tea. Beyond this Lounge was a Conference Room where we met for Lectures and Round Table discussions.

On Monday, the twenty-first, we met in the Lounge for Orientation, which lasted from nine to nine fifty A. M. and at which time we were presented to the Faculty, who are the following:

Miland E. Knapp, Clinical Assistant Professor of Physical Therapy, Director of Department of Physical Therapy.

Marian Denny, Head Technician, Department of Physical Therapy, Gillette State Hospital for Crippled Children, St. Paul.

Herman Kabat, Instructor in Physiology.

J. M. Nolte, Director, Center for Continuation Study.

William A. O'Brien, Director, Postgraduate Medical Education.

John F. Pohl, Clinical Assistant Professor of Orthopedics.

Sister Elizabeth Kenny, Special Guest Instructor, Department of Physical Therapy.

A. V. Stoesser, Associate Professor of Pediatrics.

We were given a schedule, for the days to follow, which included lectures at the Center and visits to various Hospitals. The program was laid out so as to give us a refresher course in Infantile Paralysis, which includes Myelitis, suppurative Myelitis, Acute Anterior Poliomyelitis, Landry's Paralysis (acute ascending paralysis), and Toxic Myelitis. We also had lectures on the Pathological Physiology of Poliomyelitis, by Herman Kabat. The Kenny Method was then explained to us by Dr. Pohl and Sister Kenny and we were permitted to see cases under treatment and to draw our own conclusions.

Since the refresher course of Infantile Paralysis has been ably covered by my colleague, Dr. Gregory, I will endeavor to relate to you the Kenny Method of Treatment for Infantile Paralysis, offered principally by Doctors Pohl and Knapp. Probably the best way to present their views is to use their own words which have been published by the National Foundation for Infantile Paralysis, Inc., sponsors of the course.

Dr. Pohl presented the Kenny Method of treatment by comparing it with the conventional, or Orthodox Method, which is generally known to us. On a blackboard he headed one column the Orthodox and another the Kenny Method. Under the Orthodox we find,

1. The Virus hits the anterior horn cell.
2. There is a flacid paralysis.
3. Treatment is splints in the acute stage and then re-education of muscles and physical therapy to overcome contractures.

In the Kenny Method we have the same pathology.

1. If there is sufficient destruction of the anterior horn cell, we have paralysis. (Sister Kenny does not claim results in the 15% of cases that are known to be definitely paralysed.)
2. The muscles affected present a condition of spasm.
3. The affected muscles become shortened.
4. Coordination is disorganized and incoordination frequently is seen.
5. The patient loses power in the non-affected muscles because the affected muscles are pulling the non-affected muscles from their normal resting place and retaining them in this lengthened position through the unrelaxed spasm in the affected group.
6. The non-affected muscles frequently refuse to contract due to mental alienation.

The Kenny Method can be condensed into,

1. Muscle spasm.
2. Incoordination.
3. Mental alienation.

Muscle Spasm

The term muscle spasm denotes a group of symptoms including fibulary twitching, fassiculation, hyper-ability of the muscle to stretching and a more or less tonic state of contraction of the muscle fibers which frequently cannot be overcome even by great force. Examination of patients acutely ill with Poliomyelitis bring strikingly to the attention that he suffers from something more than weakness of muscle, whether the affected muscles are painful, tender, irritable or in spasm. Probably this acute spasm in the muscle bears an important relationship to the deforming fibrotic changes in the muscles, common in the chronic stages of the disease, when inaffectively treated in the acute stage. It follows that the treatment of Poliomyelitis must begin immediately with the onset of the disease if grevious after effects are to be avoided. If unrelieved, spasm will result in the destruction of muscle tissue and cause permanent changes and deformity. It will also cause changes in the motor system by eliminating or alienating non-affected muscles and will produce incoordination in muscles concerned in joint movement. A spastic muscle becomes short and, contrary to the usual belief, is not a powerful muscle. How do we detect spasm? Principally from observation, abnormal skin creases, and so forth. The hanstrings, gastrocnemeus, back and neck are usually in spasm.

Incoordination

Incoordination, the second of the major symptoms of Infantile Paralysis is principally of two types:

1. That due to the spreading of motor impulses intended for a certain muscle to other muscles or groups of muscles due to such conditions as pain on attempted motion of the involved muscle or inability of that muscle to perform its proper function.

2. That occurring within the involved muscle itself so that ineffective contraction is produced instead of a coordinated rhythmic contraction producing maximum motion as the insertion of the muscle.

Uncontrolled voluntary motion when attempted in the presence of spasm leads to the symptoms of incoordination by the development of abnormal motion patterns and misuse of muscles. Active motion on the part of a patient unless carefully supervised and directed may do great harm, therefore, and lead to difficulties in securing the return of normal function.

Mental Alienation

Mental Alienation, the third of the major symptoms of Infantile Paralysis, is the term used by Miss Kenny to describe a condition in which there is inability to produce a voluntary purposeful movement in a muscle in spite of the fact that the nerve paths to that muscle are intact. This is a physiological block which must be distinguished from the organic interruption resulting from destruction of anterior horn cells by the disease. "Mental Alienation" may conceivably be produced in several ways, the most frequent being the following:

1. A muscle is pulled beyond its normal resting length by its opponent which is in spasm.

2. A muscle may become alienated when pain is produced in its involved opponent by the attempt of such unaffected muscle to contract.

3. The spasm, or its later results, in an affected muscle may be so severe that the braking action or check on the normal opposing muscle may discourage the latter enough to produce "alienation".

4. The disease may produce changes in the nervous system which do not actually destroy the cells or fibers but do cause loss of conduction power and interference with normal neuromuscular active.

Muscles that are nonfunctioning due to "mental alienation" may remain permanently in this state unless treated.

As an example of "mental alienation" not due to Infantile Paralysis, frequently seen by Orthopedic Surgeons, is the inability of some patients to contract the quadriceps after a knee operation or other painful lesion of the joint. Sometimes reeducation is needed for a prolonged period before normal control of the muscle is obtained in these cases.

Treatment

The Kenny treatment should be begun as soon as the diagnosis of Infantile Paralysis is made. The patient is put to bed on a firm mattress with bedboards placed beneath it. A footboard is used which extends from eighteen inches to two feet above the level of the springs, to assist in keeping the bedclothes off the patient's toes. The foot board is propped away from the end of the mattress by four inch wooden blocks. The purpose of the footboard is to allow the patient to maintain the normal standing reflexes during his stay in bed and it is not in any sense a splint. The mattress is separated from the footboard so that the heel can rest against the board when the patient is lying on the back, and so toes can project below the mattress when he is lying on his abdomen. The patient is placed in bed in a position as closely approximating the normal standing position as possible with the body straight, the arms at the sides and the legs in a straight line. A folded towel or small pad may be used under the knees to prevent hyperextension. If spasm is severe, modifications of this position may be necessary to allow relaxation of the spastic muscles. For example, with a severe spasm of the back causing hyperextension of the spine, the patient may be placed on the abodmen with a pillow under the chest and under the pelvis, or on the back with a pillow in the lumbar region to exaggerate the deformity and thereby allow relaxation of the muscles involved. If there is active spasm in the posterior calf muscles, the foot is not put in contact with the footboard until the spasm has been relaxed.

Spasm

As soon as spasm is diagnosed, treatment should be started by the use of hot fomentations which are prepared in a manner somewhat different from the usual procedure. Woolen cloths such as old blankets are cut to fit the parts accurately so that there will not be too much bulk by folding, but so that there are two thicknesses over each area to be treated. A light waterproof covering is used and around this a piece of dry woolen material of appropriate size and shape is wrapped and pinned in position. These outer coverings are spread out and placed accurately in position under the area to be treated before the hot flannel is removed from the container.

The foments are boiled and wrung from the boiling water twice through a very tight wringer at the bedside so that as much water as possible is removed. They are then applied directly and as quickly as

possible on the part so as to minimize any chance for cooling. It is well to use old wool for the wet packs because new wool seems to be more irritating to some patients. It is unnecessary to protect the skin with ointments although some patients show sensitivity to the application of wet heat by a skin rash but burns should not occur. The packs are placed accurately over the entire muscle and care should be taken to make them of adequate size since the wool tends to shrink from boiling. Joints should not be covered by the packs because of the resultant limitation of motion and increased difficulty of treatment unless the involved muscle itself cover a joint, as for example, the Deltoid covering the shoulder joint. In the hand and foot, of course, joints are necessarily covered because of the small area involved. The packs do not in any way constitute splints and they must not give the patient any sense of fixation. They are renewed usually every two hours, but may be applied as often as every fifteen minutes when the spasm is unusually acute or threatens the life of the patient, as in the involvement of the muscles of respiration. In such a case the wet packs may be used without covering. The packs are continued uninterruptedly being changed as indicated throughout twelve hours of each day. The alternate heating and cooling of the parts as accomplished by these packs seems to be the factor which tends to overcome spasm.

Passive motion through the range that can be obtained without pain is carried out at least once a day but care should be taken not to aggravate spasm by too frequent examination. The Kenny technic does not permit muscle testing or the use of respirators. Muscle testing, which incidentally has never been accurate in the acute stage of Infantile Paralysis, is definitely dangerous because of the likelihood of exaggerating spasm, or of producing incoordination and "alienation". The respirator is not used because its mechanical action in pulling on the ribs tends to aggravate the spasm in the intercostal muscles and because the treatment in the cases in which spasm as the cause of respiratory difficulty should be by hot foment. In cases in which the higher centers are involved are not benefited by either treatment, and may be definitely damaged by the use of the respirator.

Muscle Reeducation

The Kenny method of muscle reeducation is based on the symptoms and concept of the disease previously described.

First, it is assumed that nerve cells are not permanently or completely destroyed until failure of the muscles supplied by those cells to respond to treatment indicates the destruction has occurred.

Second, in addition to anterior horn cell destruction the lack of function is considered to be also the result of loss of connection with the central nervous system either through "mental alienation" by which the impulse is suppressed, or through incoordination by which the impulse is diverted to other channels, or both.

The purpose of reeducation is to restore connection of the part with the central nervous system, (to restore "mental awareness"). Muscle strength is not a primary consideration but the restablishment of "awareness" and the production of a normal rhythmic motion, no matter how weak, is the aim of the treatment. Increase in muscle strength will follow. Muscle testing in the form of attempts to determine muscle strength by isolated action of individual muscles is avoided because of the danger of producing or increasing incoordination and "alienation" as well as spasm.

The neuromuscular system is highly specialized. Although each muscle has a definite primary action, which is a direct pull upon its insertion, it rarely acts alone because of the integration and cooperation of the adjacent muscles. When a joint is moved in any specific direction a number of related muscles come into play. However, there is usually one muscle which is primarily responsible for any given motion and this muscle can be known as the prime-mover of the joint in that particular direction. One must also remember that for each motion performed by a muscle there is an opposite motion performed by an opposing muscle and that the effective function of a joint depends upon the orderly regulation of these opposing units by the controlling nervous system.

When a flexor begins to contract the extensor must simultaneously elongate in a graduated manner so that smooth action of the joint results. The elongating muscle must retain at all times a certain amount of tonus so as to be able to contract imme-

diately and reverse the joint motion. This harmonious action which allows a smooth, orderly and effective functioning of muscles is well referred to as coordination. The disappearance of a particular muscle action from the motor scheme, whether due to direct involvement of the innervation of that muscle by the disease, to indirect involvement by "alienation" or some other cause, will result in attempts by the adjacent muscles to substitute for the lost motion with the development of incoordination.

In starting muscle training this substitution of muscle action must be completely removed and prevented. Every mental and physical effort of the patient must be guided to the muscle which is being trained and to that alone. Allowing and even encouraging a patient in the free choice and substitution of muscles for the mere satisfaction of haphazard motion of the joint invites disaster.

Technic of Muscle Reeducation.—As soon as joints can be moved passively through a small range without pain or incoordination indicating that spasm is lessening, muscle reeducation within that range is started with the patient still in bed. At first this consists largely of maintaining or developing a "mental awareness" of the muscles and their insertions. Later, as spasm decreases, the more active reeducation can be added. In the presence of incorrect muscle action such as substitution or incoordination, active motions on the part of the patient are prohibited until passive motions can be carried out by the technician with complete relaxation of the patient. Having achieved this state, voluntary motions on the part of the patient may be instituted but such action is always under the control and guidance of the technician. For this later muscle reeducation the patient is placed on the treatment table in as normal a position as possible. This is considered to be the usual standing position with the arms at the side, the legs in a straight line with the body, the feet at right angles and the knees straight. The patient must be relaxed and cooperative. Reeducation cannot be carried out in babies who are crying, or in children or adults who are fearful of pain or other harm. Therefore reeducation cannot be attempted before painful spasm is eliminated.

Before treatment is started the patient is instructed to lie quiet without using any muscle other than the ones to be treated. His attention must be concentrated solely on the motion to be performed. The technician then firmly grasps the part to be treated and without causing pain carries out the intended motion passively through whatever range is possible without pain. The technician instructs the patient concerning the part to be moved by stroking the exact insertion of the muscle group to be trained. With the patient concentrating on this point of insertion the motion is carried out twice passively and then the patient is instructed to attempt to carry out that motion actively. A muscle, whether paralyzed or "alienated", which is being trained, must be made to contract at least in a mental sense if in no other way; the conscious mind must accept this "awareness" of the muscle if orderly and coordinated action is to be obtained. If any visible or palpable motion is accomplished, the treatment is stopped immediately in order to leave with the patient the memory of the accomplishment. If any muscles other than the one to be trained come into play, the attempted motion is stopped immediately and the unwanted muscles put out of action by instructing the patient to do so or by finger pressure against the muscle. Much care is observed to prevent incoordination, which is shown by this attempt of the patient to bring other muscles into play. Care is taken also not to tire the patient in any way. Treatment must be discontinued if the patient shows evidence of fatigue or is uncooperative for any reason. As the patient learns how to move the muscles, more movements are given, gradually increasing the range and number of motions, although enough work to tire the muscle is never allowed.

If there is no trace of motion in spite of absence of spasm, the proprioceptive reflexes are stimulated by placing the muscle slightly on the stretch and then stimulating the muscle through the tendon by moving the joint backward and forward. This procedure is repeated daily or several times daily even while the patient is receiving hot foments. Miss Kenny has demonstrated that function of that muscle should eventually return if the muscle can be stimulated by this procedure so that the course of the tendon can be followed from its insertion to the muscle belly. "Loss of tendon" which indicates complete loss of muscle tone is an indication of probable permanent and complete loss of function.

Sister Kenny's classification of muscles is,

1. Group One. Muscles that contract within their normal resting length.

Group Two. Muscles that have to be removed from their normal resting length before a suitable contraction can occur to perform their primary action.

Group Three. Muscle group with separate origin and common insertion and multiple action.

Group Four. Muscle groups with dual origin and dual insertion and multiple action.

Group Five. Muscles that stabilize positions obtained by other muscle groups.

The first two are the most important groups.

The biceps of the arm and the hanstrings belong to the first group and the triceps and quadriceps to the second, and one example will show the importance of this classification. The quadriceps, to contract so as to perform its normal action, must first be pulled from its normal resting position by flexion of the knee. Only then can the pull be placed at the insertion of the pattellar tendon and the knee extended. The performance of this motion is the primary function of the quadriceps muscle. So called "setting" of the muscle with the knee extended does not have any place in the Kenny method.

Muscle reeducation, then, depends upon the relief of spasm, the teaching of muscle awareness, the combating of incoordination and "alienation", and the retraining of nerve pathways back to the non-functioning muscles.

Only two positions are used, the spine and prone, and all the anterior muscles are treated before the patient is turned over to allow the posterior ones to be attended.

This outline of the Sister Kenny treatment is from lectures given by Drs. Pohl and Knapp, and writings under their names.

In addition to their lectures we were privileged to listen to Dr. Herman Kabat on pathological Physiology of Poliomyelitis. He told us that when the Sister Kenney method came to the University of Minnesota it was turned over to the Physiologists for an explanation and these gentlemen analyzing both the Orthodox and the Sister Kenny methods of treatment were forced to admit that the Orthodox treatment had no physiological worth or could not be explained from a physiological angle. While the Sister Kenny treatment lent itself admirably to such explanations, he felt that the serious symptoms of Poliomyelitis were almost entirely related to muscle and include

1. Paralysis (irreversible)
2. Physiological contracture (muscle spasm)
3. Reversible Paralysis (Mental alienation)
4. Incoordination
   a. Substitution
   b. Asynergy
   c. Arrhythmia

One of the most interesting recent developments in physiology has been the accummulation of evidence for the humoral theory of transmission at the myoneural junction. With each nerve impulse a small quantity of acetylcholine is released at the myoneural junction, and it is this chemical substance which stimulates the muscle fiber to contract. The acetylcholine causes depolarization of the membrane at this point, producing an end plate potential which acts as an electrical stimulus to the muscle fiber. This acetycholine mechanism is also responsible for excitation at parasympathetic nerve endings and at synapses in automatic ganglia.

Dr. Kabat felt that anterior Poliomyelitis produced a liberation of acetylcholine which caused a contracture. This contracture in turn caused an Ischemia, the Ischemia in turn caused pain and the pain caused a liberation of more acetylcholine completing a definite cycle, which unless interrupted would cause muscle spasm and definite permanent contracture. Another interesting feature of this study was that the hot packs of Sister Kenny have the property of breaking this cycle, by reducing the Ischemia and causing the Hyperemia. This is only one mechanism capable of explaining muscle spasm. He offers two others along similar lines. All three, he took care to say, were entirely speculative but possible. At the lectures, frequently, the whole staff participated. Sometimes they argued amongst themselves over various points at issue. Sister Kenny invariably was present and on one occasion delivered a lecture. This consisted principally of a talk concerning herself and her entrance into the field of Poliomyelitis. It has been told so often that I am not going to take up your time by repeating it.

She was at her best at the Hospital Clinics. We were fortunate in being at Minneapolis at a time when there were many acute cases of Polio. At the University Hospital we had an opportunity to ob-

serve the hot-packs and to see the cases under actual treatment. The situation there was extremely orderly. Each treatment was carried out by expert technicians, the hot packs applied and the muscular reeducation was taken care of by one of Sister Kenny's assistants. A similar situation existed at the Minneapolis Hospital. Here the conditions were not quite so ideal but we had the chance of seeing several acute cases receive immediate treatment. One case was a youngster who had paralysis of the Platysma Muscle. We were there when the child was brought in and saw his condition, with jaw drawn down and unable to swallow or to speak. We again saw the youngster after he had received hot packs for twenty-four hours and were amazed to see that he could both swallow and talk. Sister Kenny had given the child her personal attention throughout the night. One could not help but be impressed with this form of treatment. The youngsters in their acute stage being free from pain, were co-operative and lacked the apprehension that usually goes with the acute stage of Polio. Sister Kenny has mastered anatomy. She knows her muscles, their origin, insertion and particularly their connection with other muscles. She has the ability also to teach and frequently imparts this knowledge to her patients.

One patient was being examined before a group, a particularly likable negro child. Sister Kenny examined the youngster, demonstrating certain muscle groups that she wished to bring to the attention of her audience. The youngster, on being dismissed said, "Sister Kenny, you have not stimulated my Gastrocnemeus muscle." Sister Kenny replied, "And what else haven't I stimulated?" The child then immediately enumerated the various muscles which Sister Kenny had neglected to stimulate. Some people feel that this might be a case of showmanship; still other cases that we saw, while not quite so spectacular show that the youngsters obtain a considerable knowledge of anatomy which must be beneficial in their treatment.

While the results of the Sister Kenny treatment, in the acute stage, are remarkable, Sister Kenny has also obtained some good results in the older cases, although her method is primarily one for the acute case.

During the course we ran into such words as PROPRIOCEPTORS and STRETCH REFLEX, both of which have an important meaning in the Kenny technic. Kreig is responsible for the following: Functional Neuro Anatomy.

The reflex behavior falls into definite named patterns and the individual reflexes may be evoked at the will of the operator by the application of the effective stimulus. The various reflexes will now be reviewed. They should be thoroughly understood as they are the blocks from which the structure of normal postural activity are built.

An example of the simplest reflex is the KNEE-JERK. Like many other of the reflexes to be enumerated the knee-jerk can be elicited in the intact and conscious man. It can be induced by suspending the leg freely and tapping on the patellar tendon. The knee extends quickly, and as rapidly falls back to its original position. An impulse has been received by the nerve endings in the tendon, transmitted up a sensory nerve fiber, through the sensory root of the third lumbar segment, then carried by the sensory collaterals through the gray matter to the motors cells of the ventral horn, in the same segment and on the same side, and finally to the quadriceps muscle, which, contracting, extends the knee.

If the sole of the foot is stimulated by gently placing a flat surface in contact with it, the foot is pushed down on the stimulating surface. This is called EXTENSOR THRUST, because the extensor muscles of the limb are called into action. Its effect is thus the opposite of the flexor reflex. Its object is to enable a firm footing to be made. If all four feet of a mammal be simultaneously stimulated in the manner described the results is a SPINAL STANDING a condition in which the spinal animal supports itself. Spinal standing is an uncertain reaction and is not easily maintained. It will be learned later that the basal part of the brain must be present if effective support is to be obtained. Extensor thrust is diagrammed similarly to the flexor reflex, but the sense organ is a pressure receptor in the sole and the active muscles are the extensors (glutaeus maximus, quadriceps, soleus).

Still another reflex may be induced by a proper stimulataion of the sole of the foot. If a toe be clamped gently in one position the foot will be moved from side to side and the extremity flexed, obviously to free the foot. This is the CLIP REFLEX. It may be diagrammed similarly to the flexor reflex, but additional muscles: adductors and abductors must be added.

The stretching of a muscle, by moving the bone on which it acts in such a manner as to elongate the muscle, or tapping of the muscle's tendon (thereby stretching the muscle), causes that muscle to contract, and to maintain this contraction for as long as the stretching continues. This is the STRETCH REFLEX, an important and fundamental one. It can be shown to be incited by the stimulation of the specialized end-organs within the muscles and tendons. The importance of the stretch reflex and its modifications in the intact animal can hardly be overemphasized. It prevents a limb from being placed in awkward positions and gives a running start, so to speak, to muscular contraction in such movements as walking. It is "posture in the making". Normally the constant activity of the proprioceptive endings in muscles, tendons, and joints induces a state of moderate contraction or TONUS in the postural muscles throughout the body. A very minute elongation of the muscle will induce the reflex. When allowed to draw its own graph, one sees that recruitment of motor neurons is a marked trait, but that after-discharge is absent or greatly reduced. The proprioceptive receptors (PROPRIOCEPTORS) do not adapt to continuous stimuli, so the tonic nature of the contraction will continue as long as does stimulus.

The physicians course in Kenny Technic permitted us to see the clinical picture of patients suffering from anterior Poliomyelitis, requiring hot fomentations to the various parts of their bodies. We also saw the technic of muscle reeducation demonstrated. This was all carried out under ideal conditions in the hospitals in Minneapolis. During these demonstrations Sister Kenny remarked that even poor Kenny treatment was better than no Kenny treatment. Our final hospital visit was to an institution away from Minneapolis. Here the conditions were not as ideal as they were in the hospital we had visited previously due to reasons beyond the control of the hospital management. The children had received either delayed or poor Kenny treatment but in spite of this we were impressed with their improvement. I am sure that we all left Minneapolis for home feeling that Sister Kenny had demonstrated the value of her treatment for the acute cases of Infantile Paralysis.

319 Broad Street

COMBINED MEETING

RHODE ISLAND MEDICAL SOCIETY

PROVIDENCE MEDICAL ASSOCIATION

Medical Library, February 4, 1943, at 8:30 P. M.

Speakers

Dr. James E. Paullin of Atlanta, Georgia, President Elect, American Medical Association. President, American College of Physicians.

Dr. James C. McCann of Worcester, Mass. Will speak on the Massachusetts Medical Service Plan.

Dr. Herman C. Pitts. Will speak on the Rhode Island Sickness Insurance Act.

DENTISTS TO HONOR DR. GORMLY

A By-Law of the Rhode Island State Dental Society reads as follows:

> The Rhode Island State Dental Society may each year, at the annual meeting, grant its award, consisting of medal and scroll, to one who through eminent service has promoted the advancement of dentistry or furthered its public appreciation.
>
> Full authority in naming the person to whom the award shall be granted is vested in the Board of Trustees.

Under this By-Law the Trustees have voted the medal of award and scroll of the Society to Dr. Charles F. Gormly, President of the Rhode Island Medical Society. The scroll and award will be presented to Dr. Gormly at the annual dinner of the Rhode Island Dental Society on January 27.

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PETER PINEO CHASE, M.D., *Managing Editor*
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OUR OBLIGATION

At this time of War and Rationing, the Medical Profession apparently is preferred above all civilian groups; given *C* cards for extra gasoline (the doctor naming his own needs), *E* markers for the windshield, that he may drive freely in emergency, and tire permits in the earliest classification.

With such privileges come corresponding responsibilities.

If conservation of gasoline, due to tanker shortage, and of rubber, from military needs, are imperative, we doctors must freely cooperate with the lay public in effecting this conservation. Every necessary mile we drive and every mile beyond our professional requirements is so much handicap to a speedy end of the war; conversely, each *C* coupon which we can return, showing gasoline economy, is to the good in furthering this end.

Let's remember that doctors are semi-public characters, respected and observed perhaps more than the average citizen; wherefore, whether or not we like it, we are expected to set an example in all matters bearing upon the war effort. We must maintain our own homes at 65° and use due restraint in signing, for our patients, certificates for extra fuel oil; see to it that our blackout details are correct and follow the laws of rationing to the letter.

In truth, it is our especial business, here at home, to play the war game to the limit, and so to use our not inconsiderable influence in the community that others may do likewise.

ATYPICAL BRONCHO-PNEUMONIA, "VIRUS PNEUMONITIS" OR WHAT?

There is no reason to doubt that upper respiratory infections, endemic and epidemic, mild and not so mild, have afflicted the human race and its evolutionary precursors since their first appearance on the earth. Spirits, devils, miasmas, chilling the body surface with draughts and a multitude of bacterial factors have been considered and discarded as the primary etiological agents as man's familiarity with his environment has increased. Nowadays for many of these conditions an ultra microscopic virus has been found and has been shown to be associated with outbreaks of what are clinically classed as influenza and common colds.

But at present, as every practitioner knows, what seems to be a closely related but distinct disease entity prevails in epidemic, perhaps pandemic, form. This condition has been labelled "virus pneumonia" or "pneumonitis" — "virus", because attempts to isolate a virus have given rise to suggestive but thus far inconclusive results and because of its close resemblance to psittacosis, Q fever and other known virus diseases; and "pneumonia", because in many cases it is possible to demonstrate by X-ray or physical examination pulmonary involvement, usually small but sometimes extensive.

A number of pertinent questions arise. In the first place is this clinical entity new, or have we merely given a new name to a previously prevalent but unrecognized condition? All physicians who have been in general practice since 1918 will remember having seen a large number of cases of "influenza" in which, when the temperature failed to come down, or rose secondarily after the initial attack, cough persisted and vague pulmonary signs

appeared late in the disease. X-rays, when taken, showed "atypical" findings indistinguishable from those seen in the present outbreak. These cases were labelled "influenza with complicating broncho-pneumonia", "post-influenzal pulmonary infection" or given some similar designation and were generally supposed to be due to secondary infection with pyogenic bacteria—as perhaps many of them were. Again, one may ask if the disease as it now exists may be appearing in a new light because common contaminators, especially pneumococci, have been rendered relatively rare by the widespread use of sulfonamide drugs. In other words has the relative suppression of pneumococcus infections uncovered a commonly associated virus infection which now appears by itself as if it were a new condition?

These and other questions must remain unanswered until fundamental studies on the etiological factors have been successfully completed. In the meantime we may say that a condition prevails which from a clinical standpoint at least is somewhat new, which is unaffected by sulfonamide drugs, which appears to have an incubation period of nearly two weeks and whose clinical picture merges into that of influenza and "colds" with so little chance of definite differentiation in many instances as to leave the whole situation confused.

NATIONAL CONFERENCE ON MEDICAL SERVICE

1943 Meeting—Palmer House—Chicago, Illinois
Sunday, February 14, 1943

To the Editor
Rhode Island Medical Journal

The date for the 17th annual meeting of the National Conference on Medical Services has been set for Sunday, February 14. This meeting is held annually in connection with the Congress on Medical Education and Licensure, you will remember, which is scheduled this year for February 15 and 16. The place of meeting will be the Palmer House, Chicago.

We all know that the postwar period will present many critical problems to the medical profession as well as to all other lines of endeavor and that a great deal of legislation with strong socialistic tendencies will unquestionably be introduced into Congress.

In order to meet this situation, a great many feel that medicine should be better represented in Washington than it is at the present time. That there should be a permanent office there, adequately manned, so that reliable information about health can be furnished to the bureaus, Congressmen and Senators as well as the doctors back home. These men feel that this is a proposal of the greatest importance and that it should be our major topic of discussion at the meeting. We solicit your opinion either pro or con on this important matter, together with your suggestions as to people who could take part in the discussion.

Sincerely yours,

W. L. Burnap, M.D.
Secretary

UROLOGY AWARD

The American Urological Association offers an annual award "not to exceed $500" for an essay (or essays) on the result of some specific clinical or laboratory research in Urology. The amount of the prize is based on the merits of the work presented, and if the Committee on Scientific Research deem none of the offerings worthy, no award will be made. Competitors shall be limited to residents in urology in recognized hospitals and to urologists who have been in such specific practice for not more than five years.

The selected essay (or essays) will appear on the program of the forthcoming meeting of the American Urological Association, May 31—June 3, 1943, Hotel Jefferson, St. Louis, Missouri.

Essays must be in the hands of the Secretary, Dr. Thomas D. Moore, 899 Madison Avenue, Memphis, Tennessee, on or before March 1, 1943.

LESSONS FROM BRITISH EXPERIENCE IN CIVIL DEFENSE

Distributed to Regional Medical Directors by the
Office of Civilian Defense, Washington

Three years of British experience with air raids have significantly modified earlier concepts regarding the field casualty services. The following observations were made on a recent inspection of emergency medical facilities in England and Scotland.

1. Heavy raids occur invariably at night; heavier high-explosive bombs and land mines are now being employed, up to 2,000 kg., with much greater destructive effects. Incendiary bombs are used in much larger numbers, and fire is now the most serious hazard. Daylight raids are usually hit-and-run affairs in which solitary planes participate.

2. In large cities the field casualty services may handle 2,500 to 3,500 casualties during a night raid. All serious casualties are moved directly to hospitals, never to first-aid posts. Heavy raids are apt to be repeated on subsequent nights when the protective forces are exhausted.

3. A large fleet of four-stretcher ambulances is essential for life saving. Fourteen thousand ambulances were made in England and Scotland by purchasing used cars, stripping them, and then mounting a simple ambulance body on the chassis. London uses over 1,500 of such ambulances and 550 sitting-case cars. The use of tradesmen's trucks proved universally unsatisfactory; 3 out of 4 never arrived on the scene, and lives were lost due to the delay and confusion. Because of the large number of casualties to be transported in a few hours, no ambulances which carry less than 4 stretchers are employed. For the simultaneous evacuation of damaged hospitals, a fleet of 200 converted busses carrying 10 stretcher cases and 6 to 10 sitting cases are immediately available, and another 200 are obtainable within 2 hours.

4. Casualty stations (British fixed first-aid posts) are necessary at or near all hospitals and at places more than a mile from hospitals to care for minor casualties which do not require hospitalization. Many are now on a care-and-maintenance basis and are activated only during a raid. When functioning, the staff usually consists of one or two doctors, several nurses, and a variable number of aides and auxiliaries.

5. In large cities casualty stations need not be more numerous than 1 per 25,000 inhabitants; they should be located about a mile apart. There are less than 300 in the London area, with a population of about 10,000,000 and a land area more than twice that of Greater New York. In smaller, thinly settled communities, they are more numerous in relation to population, but the distances between them are proportionately greater than in metropolitan cities. Many of the minor casualties are moved to first-aid posts in sitting-case cars; some walk.

6. First-aid parties (our stretcher teams) are not necessary, are a waste of manpower, and are rapidly being eliminated. First aid at incidents is essentially a function of the rescue parties (our rescue teams), which extricate the casualties from under the debris of demolished buildings. All first-aid parties in England and Scotland are, therefore, being merged into the rescue parties. They include a leader, an assistant leader, and eight other members, and are entirely independent of the fire department. They are a life-saving service related to the medical services concerned in field casualty work.

7. The experiences of Britain under air-raid conditions have dispelled many preconceived notions, concerning first aid. Almost all raids occur at night; the victims are crushed under the debris of demolished buildings and are either dead or severely injured; less than a third are slightly injured and can be cared for at casualty stations; all the severely injured must go to a hospital; victims are invariably covered with dust and dirt which hangs in the air for hours. The conditions under which the rescue workers encounter the injured beneath the structural debris, the darkness and the dust which always fills the air, the large proportion of dead and severely injured, and

the urgent need for immediate hospitalization make it impossible to apply most peacetime concepts of first aid.

8. Wounds are usually grossly contaminated and need only be covered with a shell dressing until the casualty reaches the hospital. Hemorrhage is usually controllable with a pressure dressing. The tourniquet is rarely employed. Burns are covered only with sterile gauze until the casualty arrives at the hospital. Tannic-acid jelly as a first-aid dressing for burns has been discarded because of the dirt which invariably contaminates the burned surface, because the jelly deteriorates rapidly, and, lastly, because tannic acid ignites in the presence of phosphorus when applied to burns caused by the explosion of phosphorus-oil bombs.

9. Traction splints are not used. An exception is made if the casualty must be transported a long distance over country roads. Unlike Army field experience in the last war, the few miles of travel to a hospital over the paved roads of a city do not warrant the application of traction, especially as the darkness and the conditions of an air raid also make hurried application of the procedure difficult or impossible. All that can be done is to place the fractured extremity gently in alignment, bind it with triangular bandages to the uninjured leg or to an improvised splint, or apply a Thomas splint if one is on hand. Movement of the fragments can also be minimized by snug application of the blankets according to the Wanstead technic of blanketing and by the use of sand bags, which should always be carried in the ambulance.

10. Shock is treated at the incident by prompt administration of adequate doses of morphine (up to ½ grain for adults), coramine, proper blanketing, administration of fluids, and the use of hot-water bottles during transportation to the hospital. The use of plasma or blood transfusion is deferred until arrival at the hospital; it is ordinarily quite impossible in the darkness, dirt, and confusion at the incident.

11. The presence of a physician at the incident is invaluable, but more than one is unnecessary. In fact, one physician may cover several nearby incidents, leaving his nurse or one of the nursing auxiliaries of his emergency team at the incident while he moves temporarily from one to another in the immediate neighborhood.

12. Even though a single night's casualties requiring hospitalization may total one or two thousand, large hospitals rarely receive more than 50 to 100, the load being distributed as evenly as possible throughout the city.

13. A large casualty receiving hospital is often related to one or more peripheral hospitals in the suburbs or in a country district. There are now four base hospital beds for each casualty bed in the cities.

14. Upon receipt at a local report and control center of a message from an air-raid warden that an incident and casualties have occurred, an "express party" is immediately dispatched to the scene. An "express party" includes one rescue-first aid party, one ambulance, one sitting-case car, and one mobile medical unit (our mobile medical team). The latter consists of one physician, one nurse, and two auxiliaries. No other equipment and personnel of the emergency medical service is dispatched unless additional assistance is requested by the incident officer (usually a higher police official) or by the incident physician on the scene. In this manner useless movement is avoided and equipment and personnel of the community is carefully conserved.

PROVIDENCE MEDICAL ASSOCIATION

The 96th Annual Meeting of the Providence Medical Association was held at the Medical Library on Monday, January 4, 1943. In view of the large attendance of guests invited to hear the discussion on infantile paralysis the scientific program was presented early in the evening, followed later by the business session which was restricted to members of the Association.

Dr. Kalei K. Gregory presented a paper on "Acute Anterior Poliomyelitis — A Review of Essential Features", after which Dr. William A. Horan spoke on "Some Impressions of the Kenney Treatment". These talks were followed by a demonstration of application of fomentations given by Miss Helen Cahalan and Miss Madeline Bailey. The orthopaedic viewpoint was briefly discussed by Dr. Herbert E. Harris, and the pediatric viewpoint by Dr. Banice Feinberg.

In his presidential address Dr. Henry E. Utter highlighted the work of the Association during the past year, and forecasted some of the future problems of medicine in the light of present social trends.

Following this oration Dr. Utter called for nominations for President of the Association for 1943, and Dr. William P. Buffum moved the nomination and unanimous election of Dr. Emery M. Porter. The motion was adopted and Dr. Porter was declared elected, and was escorted to the rostrum where he spoke briefly on plans for 1943, and announced that his complete list of committee appointments would be announced in the February issue of *Medical News*.

A motion being in order for the nominations of a Vice President, Dr. William M. Muncy moved that the entire slate of officers as recommended previously by the Executive Committee be declared nominated and be elected. The motion was unanimously approved, and the Secretary cast the ballot electing the following slate of officers for 1943:

Vice President ..... Albert H. Jackvony, M.D.
Secretary ..... Frank W. Dimmitt, M.D.
Treasurer ..... Herbert E. Harris, M.D.
Executive Committee (2 members for 5 year terms) ..... Henry E. Utter, M.D.; Frank I. Matteo, M.D.
Councillor to R. I. Medical Society (2 year term) ..... John G. Walsh, M.D.
Trustee of R. I. Medical Library (1 year term) ..... Peter P. Chase, M.D.

Delegates to the House of Delegates of the Rhode Island Medical Society

John A. Hayward, M.D.	Robert H. Whitmarsh, M.D.
Harry C. Messinger, M.D.	Joseph L. Belliotti, M.D.
Charles L. Southey, M.D.	George W. Waterman, M.D.
Henry F. McCusker, M.D.	Jerome J. McCaffrey, M.D.
James Hamilton, M.D.	Frank W. Dimmitt, M.D.
John G. Walsh, M.D.	Louis A. Sage, M.D.
Merle M. Potter, M.D.	Gordon J. McCurdy, M.D.
James H. Fagan, M.D.	Edward S. Cameron, M.D.
Kalei K. Gregory, M.D.	Bertram H. Buxton, M.D.
Raymond F. Hacking, M.D.	Harmon P. B. Jordan, M.D.
Ralph Di Leone, M.D.	Harold G. Calder, M.D.
Joseph B. Webber, M.D.	Anthony V. Migliaccio, M.D.
Frank J. Honan, M.D.	A. Henry Fox, M.D.
Arcadie Giura, M.D.	

Dr. Herbert E. Harris, treasurer, read his annual report for the year, which was approved and placed on file.

The new Secretary, having been escorted to the Secretary's desk, presented the report of the Executive Committee making the annual recommendations of appropriations and setting the annual assessment of dues for 1943 for active members at $15. The report of the Executive Committee was unanimously approved.

The Secretary reported the recommendation by the Executive Committee of Dr. Henry E. Darrah for membership in the Association. On the motion of Dr. William M. Muncy the election of Dr. Darrah was made unanimous.

The President announced the receipt of the obituary tribute to the late Dr. Charles N. Raymond which had been prepared by Dr. Herbert Armington and Dr. Edmund D. Chesebro.

The President announced that reports of Committees would be published in the next issue of *Medical News*, and he then called for adjournment, at 11:20 p. m., with a rising vote of appreciation for the services rendered by Dr. Utter as President of the Association during 1942.

RECENT BOOKS

THE MIND AND ITS DISORDERS. By James N. Brawner, M.D. Medical Supt., Brawner's Sanitarium, Smyrna, Ga. Cloth. Price $3.50. Pp. 228. Atlanta, Ga. Walter W. Brown Publishing Co. 1942.

This book is an excellent practical handbook of Psychiatry with a modicum of neurology added to make a complete picture. It is written simply, with a minimum of psychiatric clichés which are so irritating to the ordinary medical man. It covers very well a wide field and could be included in the general practitioners' library with profit.

It is not a reference book and in no sense of the word is it meant to be an authority on any one psychiatric picture. It is not an encyclopaedia nor was it written to be.

Dr. Brawner deserves praise for having gotten together an admirable, simply written, general summary of the field of psychiatry. I heartily recommend it to the attention of the average medical man whose knowledge of psychiatry is usually lamentably small.

GEORGE L. YOUNG, M.D.

THE HAND, by Condict W. Cutler, M.D., W. B. Saunders Co.

This is a comprehensive, excellent monograph on probably the most important subject in the realm of surgery. The one thing which gives man his great advantage over the so called lower animals is the elaborate versatile piece of mechanism at the distal end of his upper extremity. His much vaunted intellect would be of little service to him could he fashion none of the tools which build his civilization or even grasp a club to make him more physically equal to the powerful brutes.

And yet in our hospitals hand surgery is frequently left to the interns, while the august visiting men devote their skill to saving for a little longer suffering a pindling percentage of stomach cancers or to obviating a limp which a poorly handled lower leg fracture might give its owner.

A short section at the beginning is devoted to summarizing the high spots of the intricate anatomy without a detailed knowledge of which accurate surgery cannot be done.

The next 80 pages deal with acute infections, the remarkably specialized treatment of which Kanavel put on a firm basis over a quarter century ago. We have seen many otherwise competent surgeons who we felt had not deigned to master this detail.

After this are 75 pages on Specific and Chronic Infections with a bibliography of 42 articles. Some of this looks like padding. It is not demonstrated that there is great peculiarity in the hand as regards gas gangrene, tetanus, anthrax, glanders, rabies or lupus for instance.

The following few chapters on burns, contusions, wounds, etc., have some excellent points especially a fairly elaborate discussion of the surgery of tendons.

Fractures, not an elaborate subject when the hand only is considered immediately become of great interest in the carpus and forearm. The Jews speak of the "fuss" when they get below the groin. Dr. Cutler commits a Hebraism when he brings the hand so high. But his chapter on amputations and compound fractures is a good one.

The remarkable modern work on repair and construction by Koch, Iselin, Bunnell and others is touched on lightly but yet in a suggestive and admirable manner as is the related subject of deformities.

"Tumors" presents little particularly characteristic of the hand but the final chapter on Constitutional Diseases shows interestingly how these manifest themselves in the hand.

Despite some minor matters open to small criticisms we consider this a good book and general surgeons who expect to work on the hand should have access to it.